Social Impairment and Mental Health

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Abstract

Stress resulting from living in large cities (and alienation in certain individuals) fuels the high incidence of psychiatric illnesses in modern metropolises. Some pathology as Schizophrenia and Asperger has deficits on social cognition mechanisms and its presentation may be influenced by social environmental impact. Even more, common mental disease such as depression and personality disorders has a great impact by environmental social deficits and present poor social cognition performances on some specific areas. The purpose of current brief review is to provide an overview of the mechanisms who impacts on social relationships (social cognition) and the effects of social deprivation in people with mental illness.

Keywords: Social cognition; Stress; Schizophrenia; Psychiatric illnesses; Depression

Introduction

Social interaction is a behavior that appears early on primates and is related with changes on neo-cortex [1, 2]. In human, first social interaction is attachment: the behavior of achieve or maintain proximity with another person considered stronger [3]. It motivates the search for proximity between the child and their parents and promotes social bonding and care behavior [4]. The main characteristics of the caregiver are the sensitivity to emotional cues in response to signals from the child [5, 6]. Newborn can imitate adult gestures [7] and facial recognition of baby's emotions activates areas of empathy (insula) [8, 9] and reward (orbitofrontal cortex) on mother [10, 11] and child [12]. So, from first interactions humans are exposed to social behavior and require social skills to interact with others, using the social cognition mechanisms: empathy, reward learning, imitation, tracking intentions, mentalizing, theory of mind and meta-cognition [13]. Impairments on social skills and social cognition mechanisms may produce intense effects on social performance and would be one of the factors of important disability on mental illness. Our brief review point to understand how social cognition impairments may be part

of mental illness and how social environmental deficiencies may impact on development of mental illness.

Review

Stressors resulting from living in large cities (particularly the alienation in certain individual experiences) fuel the high incidence of psychiatric illnesses seen in modern metropolises, where the incidence of schizophrenia, for instance, is twice that of rural environments [14]. Social cognition is one of the main areas of impairment in schizophrenia [15]; areas such as facial emotions processing [16, 17] and understanding of other people's mental states could significantly affect the way of interacting with other people and society [15]. Recent studies have shown abnormal global functional connectivity density - increased connectivity in the subcortical regions and decreased in cortical regions- [18] related with "social brain" clusters (fusiform gyrus, left lingual gyrus, left middle and inferior occipital gyrus) during empathy tasks [19]. At electrophysiological level, significant reduction of attention markers on memory and perception tasks [20, 21] may be involved on poor "top-down" regulation of subcortical regions involved on social reaction – i.e., amygdala- [22]. Those findings have confirmed the interest of psychopathologists in studying the "lack of common sense" and "loss of vital contact with reality" [23, 24]. Despite evidence, it is still neglected from the psychometric assessment in schizophrenia, as in MATRICS (Measurement and Treatment Research to Improve Cognition in Schizophrenia) [25, 26]. This is an important area for future research, considering that poorer interpersonal functioning (family and peers) has predicted higher levels of bizarre experiences and persecutory ideation [27] and some studies have shown how one third of schizophrenic patients feel socially isolated (or partially isolated) at age of 13-14 years (previous to first episode) and post first hospitalization [28]. Thus, they manifest high levels of social restriction versus control group (just 15% belong to a social organization or frequent their friends once on a week versus 41-53% of control group) when they return to the community [28]. Systematic reviews about epidemiological studies have indicated that the rate of schizophrenia and related disorders is affected by some environmental factors: the association with urbanisation and some ethnic migration might indicate a common environmental influence linked to chronic experience of social disadvantage and isolation [28, 29]. Insofar, separation and

death of a parent before the age of 17 years were strongly associated with 2- to 3-fold-increased odds of first episode of psychosis [30]. As discussed, schizophrenia is an illness with four different levels of "social phenomena" interactions (social cognition, social brain, social behavior and social functioning) [14].

In autism spectrum disorders and Asperger syndrome, qualitative difficulties in social interaction are present [31]. Asperger patients have shown high variability in the social cognition neuropsychological tasks: mostly subnormal performance in emotional processing, theory of mind, empathy, moral judgment, social norms and self-monitoring [32, 33]. Difficulties in the use of nonverbal behaviours to regulate social interactions – such as reading the emotions in eyes- are present [34, 35]. Those deficits produce a pattern of decreased ability to implicitly encode and integrate contextual social information without correlation with executive functioning [32, 36]. All those impairments are translated in a poor clinical performance in social area (failing in developing interpersonal relationships, difficulties in sharing and expressing pleasure with people, and lack of social-emotional reciprocity or social judgment) [37]. Neurophysiological studies have shown how blunting of "event related desynchronisation" (on α and β band) is associated with impairments in imitation tasks [38]. Suppression of that band of frequencies ("Mu suppression") is important when participants are engaged in a social game [39]. Abnormal activation of amygdala, superior temporal sulcus, frontal girus and orbitofrontal cortex may be related with abnormal gaze detection on its communicative and social value [40].

Depression is strongly linked to social isolation along the vital cycle with statistical and bidirectional interaction between development of the disease and social isolation [41]. Antenatal depression is associated with poor social adjustment and less social support, social exclusion and migrant social isolation [42, 43] and postpartum depression with poor social adjustment, less social support and less involvement of the baby's father [42,44,45]. Prevalency of affective disorders in childhood is 2.91 to 3.51 times greater when children are not living with parents [46]. Also, have been observed important disruptions in academic, social, and familial functioning in depressive adolescents [47]. Increased connectivity between prefrontal cortex (PFC) and amygdala is present in adults with depressive episode and history of childhood trauma during affective tasks [48] and increased connectivity has been showed in suicidal-depressed adolescents on left cerebellum and the left lingual gyrus [49]. In adulthood, several studies show how loneliness between 50-68 years affects the development of depressive symptoms and predicted subsequent changes in depressive symptomatology. Subjective isolation is related with increase mortality risk over a 6-year period, depressive symptomatology and higher blood pressure. Also, loneliness is related with daytime dysfunction and sleep fragmentation. The hypothesis is that the brain has evolved to monitoring the status of one's "social body": loneliness serves as a signal to draw attention to threats or damage to one's "social body" [41]. Today, new approaches are focusing on studying poor empathic abilities as a potential cause of poor social skills in patients with major depression [50]. Greater number of past depressive episodes is related to less "perspective taking" ability [51] and depressive episode is correlated with increased "personal distress" scores [52]. On neurophysiologic performance depressed people seem to exhibit disturbed P300 component (reduced and slower P300 for happy faces) [53] and, faster P3b in individual with anxious tendencies [54]. An affective negative bias is observed: they can't avoid attending to negative information in their environment [55] and has higher P1 and P2 amplitude for sad faces compared with other faces [56].

Surprisingly, borderline personality (BPD) patients identifies significantly better than controls emotions on the "Reading the Mind in the Eyes Test" for both 'neutral' and 'positive' expressions [57]. Depression and rejection sensitivity partially mediates the relationship for neutral stimuli [58]. This findings contrast with the poor performance of BPD patients on others theory of mind tasks, such as "Faux pas" (a story set were patients must to discriminate social mistakes) [59], suggesting a poor judgement performance in social tasks. In fact, individuals with BPD exhibit a negative bias to appraise trustworthiness in facial stimuli and ambiguous trust decisions are associated with longer response times (but this bias is not present when evaluating emotions like fear) [58]. BPD patients show different level of increased activity in the amygdala when viewing emotional faces (Ekman faces) even in neutral faces. Affective instability is correlated with greater amygdala responses and less Inferior Frontal Gyrus (IFG) recruitment [60]. ''Is still matter of discussion if executive attention has an impact on BDP appraisal of social stimuli''. They have slower responses in orienting and conflict resolution between congruent and incongruent stimuli on attention network task [61]. A relationship has been established between re-orienting attention responses and Theory of mind'' [62].

Conclusion

Social impairments and social isolation is an important area for future research and development of policies of mental health. Cognitive neuroscience is providing contemporary neural system models for understanding psycho-social factors and epigenetics offers novel insights into how social context and environmental factors translate into biological changes at the level of gene expression [63]. New discoveries in human neuroscience must be adapted for the majority of psychiatrists and the new knowledge is essential for assessment and treatment planning [64]. The objective is not to stigmatize the absence of social skills [65], but to recognize that human beings are the product of some social relations and his ability to manage himself and his ties with activities that give recognition [3]. Some treatments such as assertive community treatment and peer support have been developed to improve these impairments on social network for patients with severe mental illnesses [66-69]. The future is to develop new pharmacological and non-pharmacological treatments and consider as a main issue of concern all the social impairments on our clinical approach with patients with mental illness.

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